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Repurposing Approved Drugs for Chemoprevention: Mechanistic Insights and Clinical Prospects

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ABSTRACT

In this paper, perspectives on the use of approved, well-characterized drugs as chemopreventive agents to prevent cancer incidence among identified risk populations will be discussed. The background adds that pleiotropic agents, such as metformin, aspirin/NSAIDs, statins, selective ER modulators, bisphosphonates, and antivirals, are agents that overlap with carcinogenic pathways with tolerable doses and known safe profiles. The rationale is to combine the mechanistic evidence with trial evidence to determine what drug-risk combinations result in net benefit and how biomarkers can be used to inform it. The anticipated outcomes are identification of mechanism-based actionable mechanisms (AMPK activation, COX-2 inhibition, HMG-CoA-mevalonate pathway blockade, hormonal pathway modulation, anti-viral oncoprevention), definition of requirements to prioritize the candidates (effect size, toxicity, interaction with exposures, cost), and a description of responsive phenotypes by molecular risk markers, minimal residual disease signals, and polygenic/clinical scores. The paper envisions the proposal of adaptive trial designs, use of pragmatic endpoints (precancer regression, validated intermediate biomarkers), drug-diet synergy concepts and pharmacovigilance models that are appropriate to long-term prevention. It will map regulatory and implementation strategies with a particular focus on real-world evidence networks to promote rapid adoption in cases where benefit-risk and value are positive.

KEYWORDS: Drug Repurposing, Chemoprevention, Metformin, Aspirin, Statins, Selective Estrogen Modulators, Cox-2, Ampk, Biomarkers, Adaptive Trials.

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INTRODUCTION

The "drug repurposing" approach for "chemoprevention" is a promising approach to decreasing cancer risk for susceptible populations. Several "well-characterized drugs", including "metformin", "aspirin", "statins" and "selective estrogen modulators" have been reported to show overlapping activity with key carcinogenic pathways. Mechanistically, this reflects the ability of established therapies, such as 'COX-2' inhibition and 'AMPK' activation to overcome tumor-initiating events. Integration of "biomarkers" allows for precise identification of these responsive phenotypes and facilitates assessment in so-called "adaptive trials." Such an approach strikes a balance among efficacy, safety and long-term tolerability while considering cost and accessibility. "By bringing mechanistic insights together with evidence from clinical trials, repurposed agents can be used to inform effective prevention strategies and promote real-world implementations of cancer risk reduction."

PROBLEM STATEMENT

Despite progress in cancer therapy, incidence and mortality continue to be high and prevention is not yet widely used. Conventional chemopreventive strategies such as vitamins, phytochemicals, and dietary interventions have yielded inconsistent results due to variable bioavailability, poor efficacy, and poor translation to clinical success [1,2,3,4]. In the present literature, the identification of agents with established mechanisms of action, acceptable safety profiles, and long-term applicability [5,6,7] still pose considerable challenges. Therefore, there is an urgent need to investigate "repurposing of approved drugs" as chemopreventive agents, especially drugs such as metformin, aspirin and statins, that specifically target essential "signaling pathways" involved in the development of cancer [8,9,10].

RESEARCH SIGNIFICANCE

The latter is important as the work will bridge the gap from mechanistic to clinical relevance for cancer prevention. Unlike novel agents, drugs with approved status contain known pharmacological and safety data, which makes these drugs suitable candidates

for fast-tracking into preventive strategies [8,11]. Repurposing NSAIDs, statins, and selective estrogen modulators are examples of agents with the potential to modulate oncogenic pathways, reduce precancerous progression, and have improved cost-effectiveness in high-risk groups [5]. This strategy overcomes limitations of previous chemoprevention studies by taking advantage of precision biomarker-guided approaches, adaptive trial design, and real-world feasibility. At the end of the day, learning drug-pathway combinations that are actionable can lead to a dramatic reduction in cancer burden, while accelerating regulatory approval and adoption.

LITERATURE REVIEW

Cancer chemoprevention research is increasingly focused on repurposing approved drugs as well as natural compounds to target oncogenic signaling with safer long-term approaches. Ren et al. [8] point out that pathways for tumor initiation include PI3K/AKT/mTOR, MAPK and NF-kB and that agents such as metformin (activates AMPK) or aspirin (blocks COX-2) have strong mechanistic rationale and good safety profiles. This mechanistic specificity enables coupling to biomarker-guided risk-specific prevention. Tuli et al. [12] also support the idea that drug repurposing can be complemented by immune-based mechanisms, such as checkpoint modulation and cytokine signaling, which could be synergistically inhibiting early tumorigenesis using immunotherapeutics and agents such as NSAIDs or statins. Tratnjek et al. [2] present evidence from bladder cancer studies that nuclear receptor signaling by vitamin A and retinoids regulates apoptosis and differentiation, but clinical use is hampered by toxicity and variability issues. Siddiqui et al. [13] expand this framework in prostate cancer with the study of phytochemicals such as resveratrol and fucoidan, which modulate oxidative stress and androgen pathway, offering preclinical evidence for reduced tumor growth, though with little clinical validation. Similarly, Mubeen et al. [14] highlight multi-target phytochemicals like quercetin and EGCG with Nrf2 and NF-kB targets to curtail the oxidative DNA damage with enhanced clinical feasibility by novel formulations. Collectively, these studies show that mechanistic knowledge from repurposed drugs, bioactive compounds and immunomodulation provides a promising, multi-layered approach, toward effective chemoprevention. Identifying the best strategies for translating mechanistic promise into population-level cancer prevention outcomes requires research that includes adaptive trial designs, biomarker stratification, and synergistic drug-diet strategies from the literature.

RESEARCH METHOD

This secondary method of this research extracts literature-based data from published ecological preclinical and clinical studies of repurposed drugs for "chemoprevention" and epidemiological arms of these studies. This framework allows for the assessment of combinations of drug risks without the need for conducting new trials, saving time and resources and maintaining an approach that is ethically feasible. Thematic analysis is used to determine recurring underlying patterns of mechanistic pathways, biomarker responses, and clinical outcomes across studies. Themes including "COX-2 inhibition," "AMPK activation," and drug-diet interactions are coded and synthesized. This approach is justified because it offers a principled way of combining heterogeneous evidence, highlighting actionable mechanisms, and informing future adaptive trial designs for cancer prevention.

RESULT AND DISCUSSION

Mechanistic Pathways Targeted by Repurposed Drugs

Repurposed drugs have chemopreventive potential owing to the fact they modulate carcinogenesis-related molecular pathways with well-characterized pharmacodynamics. According to Spendlove et al. [15], pathway-based repurposing frameworks like Pathway2Targets identify key oncogenic nodes, including PI3K/AKT/mTOR, JAK/STAT and NF-kB, where existing drugs can exert an effect. For instance, metformin activates AMP-activated protein kinase (AMPK) and thereby suppresses anabolic tumor-promoting metabolism by downregulating mTORC1 signaling. Selective COX-2 inhibitors and aspirin inhibit the production of prostaglandin E2, thus restricting proliferation induced by inflammation. Hernandez-Lemus and Martinez-Garcia [16] highlight translational bioinformatics for prioritizing drug-target interactions, exhibiting how statins block the mevalonate pathway to inhibit HMG-CoA reductase and thus hamper prenylation of RAS and RHO GTPases required for malignant transformation.

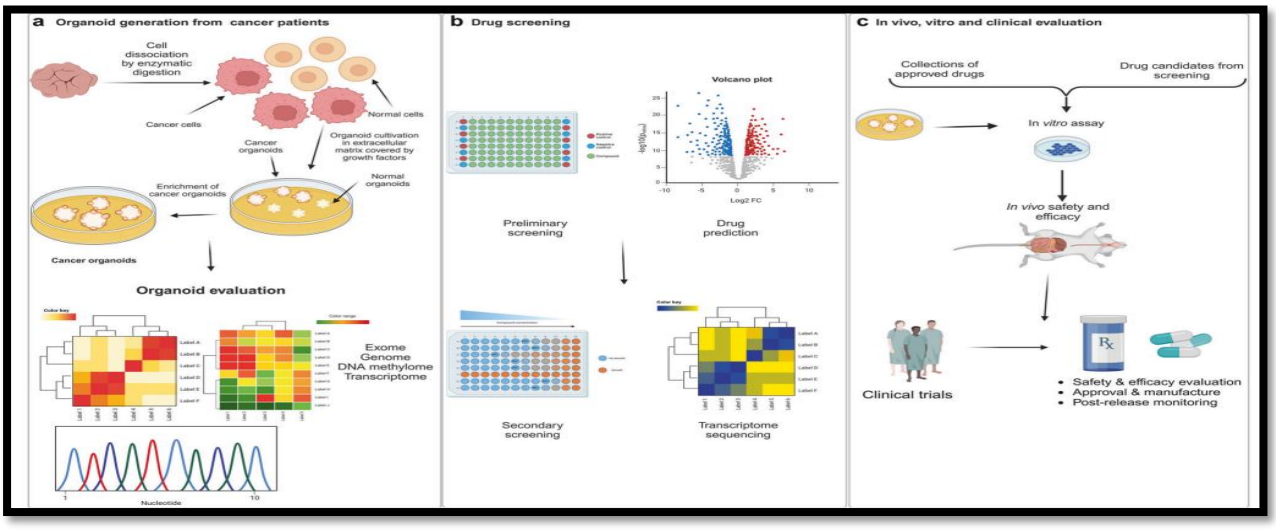

Figure 1: Tumoroid model illustrating patient-derived organoid generation and drug repurposing candidate screening for testing.
(Source: [17])

Similarly, selective estrogen receptor modulators (SERMs) modulate ERa-mediated transcription and thereby modify hormonal signaling that is at the core of breast and endometrial tumorigenesis. Xia et al. [17] remind us that antiviral agents such as ribavirin and acyclovir, though they are mostly poised for viral replication, also overlap with oncogenic processes by attenuating virus-driven oncogenesis in HPV or HBV-associated cancers. These mechanistic intersections provide evidence as to how currently available drugs target hallmarks of cancer including sustained proliferative signaling, resistance to apoptosis, and inflammatory microenvironment formation. Taken together, the selection and targeting of AMPK, COX-2, HMG-CoA, ERa, and virus replication pathways not only demonstrates the accuracy and translational potential of drug repurposing for chemoprevention, but also supports its position in the risk-adapted cancer prevention paradigm.

Clinical Evidence Supporting Chemopreventive Applications

Clinical studies provide evidence of measurable chemopreventive efficacy that is associated with validated biomarkers and risk reduction outcomes for repurposed and natural agents. In the domain of skin cancer prevention, Tow et al. [18] note strong evidence of topical diclofenac, a COX-2 inhibitor, leading to significant regressions of actinic keratosis, and of nicotinamide supplementation, which reduced non-melanoma skin cancer incidence in high-risk cohorts by 23%. Boretti [19] reports clinical validation of aspirin, which is shown to reduce colorectal adenoma recurrence dose dependently through suppression of COX-2 and prostaglandin synthesis. Statins have been shown to have protective effect in hepatocellular carcinoma patients with chronic hepatitis B and C by interfering with the mevalonate pathway and inhibition of oncogenic prenylation pathways.

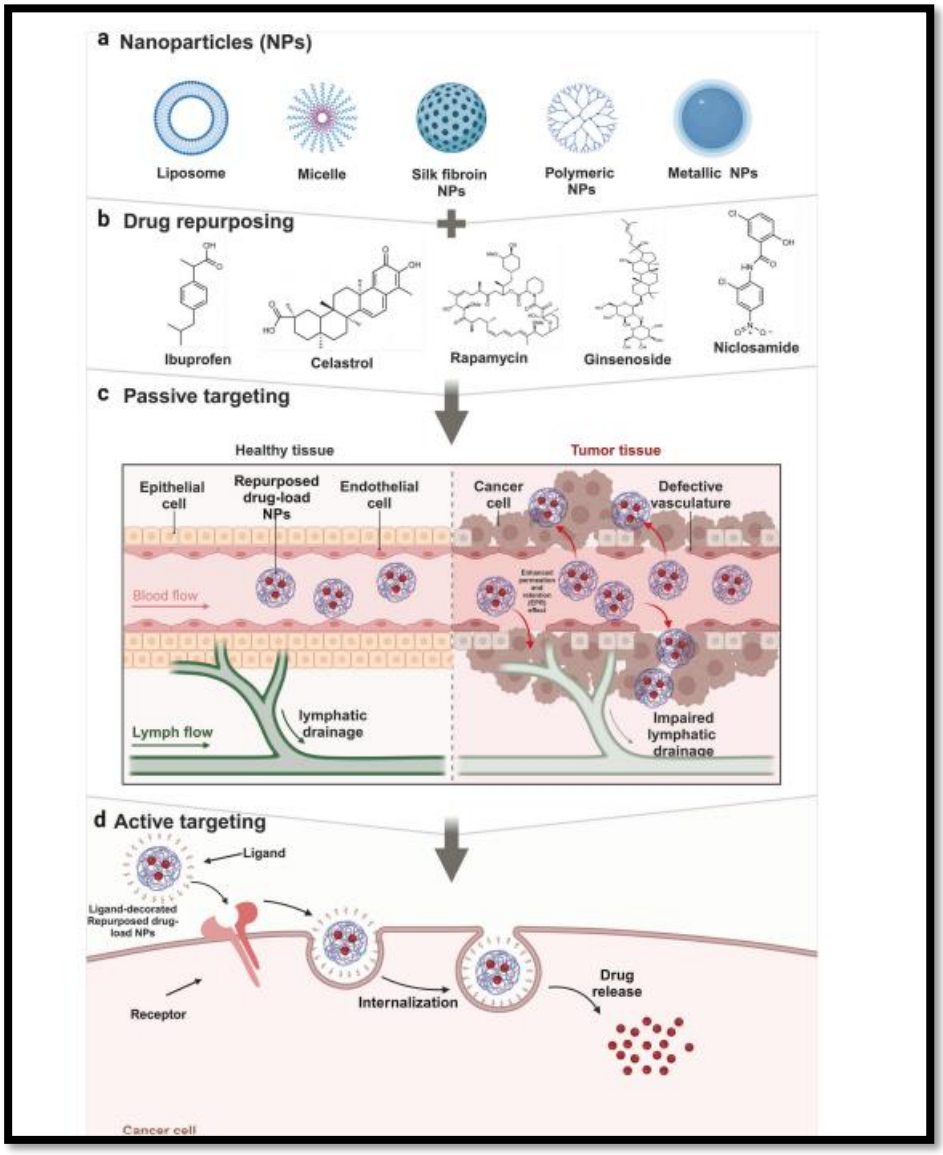

Figure 2: Nanocarriers enabling targeted delivery of repurposed cancer drugs through diverse active and passive mechanisms.
(Source: [17])

Swetha et al. [20] support the translational value of phytochemicals by highlighting curcumin's ability to modulate NF-kB and Nrf2 signaling, which has translated to objective reductions in oral leukoplakia progression in early phase clinical trials. Similarly, green tea polyphenols (epigallocatechin gallate [EGCG]) were shown to reduce the occurrence of prostate intraepithelial neoplasia in high-risk males. These clinical results confirm that the mechanistic targeting seen in preclinical models is translated into decreased progression of premalignant lesions and incidence of early-stage cancers. Taken together, the data from randomised

controlled trials and observational cohorts show a clear chemopreventive effect of agents such as aspirin, statins, nicotinamide, and phytochemicals, and helps us to consider repurposing drugs as part of precision prevention strategies.

Role of Biomarkers in Identifying Responsive Populations

Biomarkers offer important tools to stratify populations that would benefit from chemoprevention. Passaro et al. [21] point to indications such as circulating tumor DNA (ctDNA), minimal residual disease (MRD) signals, and polygenic risk scores as predictive indicators for chemopreventive efficacy that would allow for patient-specific drug allocation. For example, the response to aspirin in colorectal cancer prevention is related to PIK3CA mutations, whereas the efficacy of statins is associated with HMGCR expression signatures. Han et al. [22] show that a metabolic stimulus-sensitive marker such as Ajuba, a LIM domain protein that governs cytoskeletal dynamics, responds to the metabolic stimulus and could be used as an early predictive biomarker for treatment efficacy.

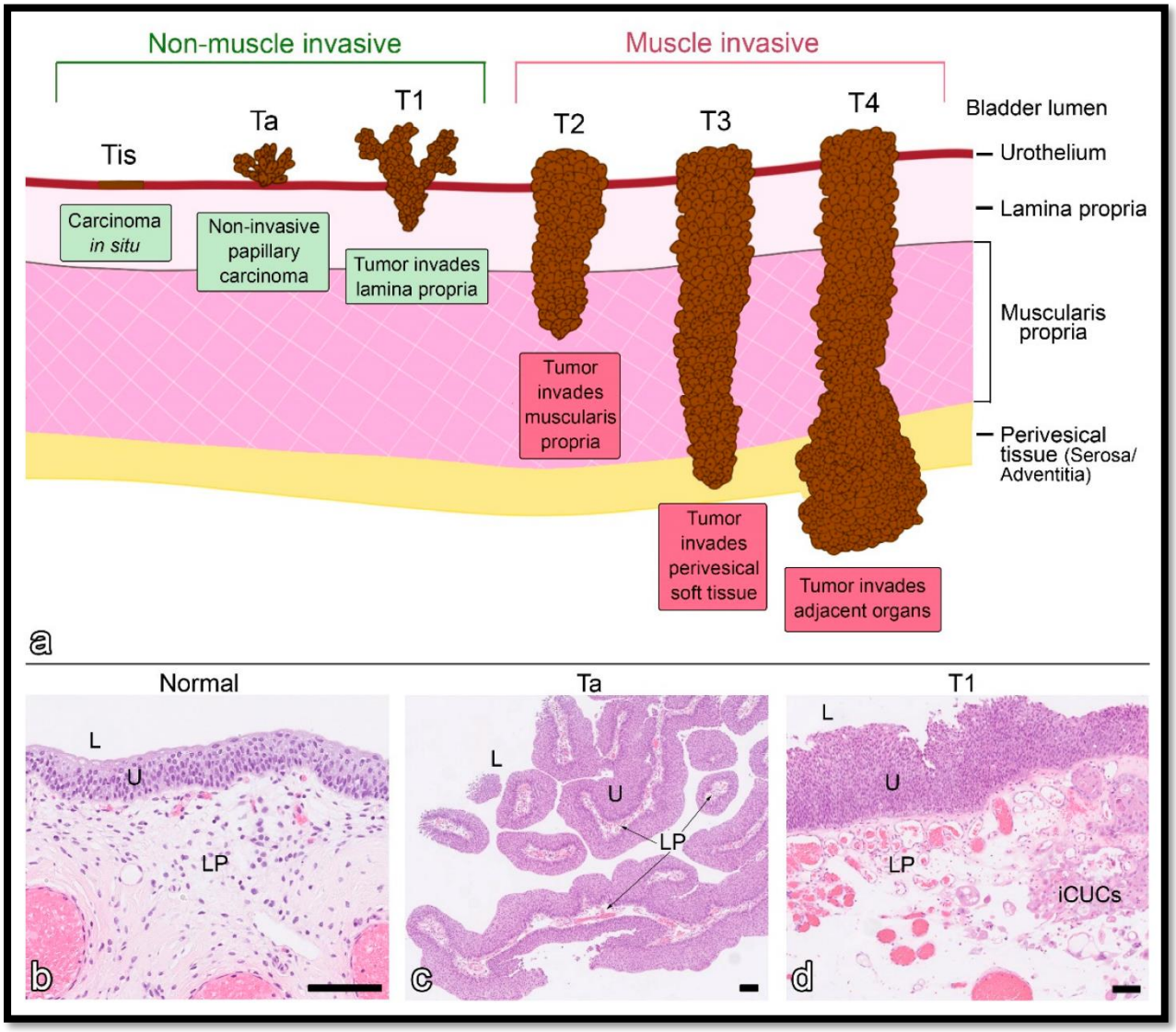

Figure 3: Staging of bladder cancer by TNM system with histological sections of normal, non-muscle invasive, and invasive tumors.
(Source: [2])

Lemos [23] focuses on the translation of biomarkers from the gene-level stress response to population-level uses; highlighting that the expression of NF-kB and Nrf2 targets implies the neutralization of cellular oxidative stress as a characteristic of preventive drug action. Scheid et al. [24] in the context of epilepsy demonstrate how electrophysiological biomarkers can be used to predict treatment response prior to therapy, which in principle is directly applicable to CCP where early biomarker changes (e.g., Ki-67 reduction in premalignant lesions) can predict long-term outcomes. Together, biomarker-enabled frameworks enable clinicians to detect responsive phenotypes, to track drug-diet interactions, and to confirm intermediate endpoints such as regression of actinic keratosis or oral leukoplakia. These results validate biomarkers as a critical element of precision chemoprevention to ensure optimal drug-pathway alignment and to reduce unnecessary exposure.

Prospects for Adaptive Trials and Real-World Integration

Adaptive trial design is considered a paradigm shift in chemoprevention by virtue of allowing simultaneous adaptation of the protocol based on biomarker dynamics and intermediate efficacy endpoints. Daca-ALvarez et al. [25] Master protocols and

platform clinical trials that concurrently assess multiple treatment agents, including COX-2 inhibitors, SERMs, and statins in risk-stratified cohorts defined by molecular anomalies including KRAS, PIK3CA, and TP53 mutations. Edsjo et al. [26] ethically pose the integration of precision oncology's concepts where liquid biopsy assays picking up signals of serum circulating tumor RNA (ctDNA) and minimal residual diseases (MRD) act as surrogate endpoints for adaptive dose controlling.

Arzika et al. [27] achieve proof of concept for large population adaptive cluster randomised platform trials, and illustrate the use of modular design methodologies for faster assessment of community-based interventions. This model can be used for cancer prevention to use pragmatic endpoints such as regression of actinic keratosis, oral leukoplakia, or intraepithelial neoplasia of the cervix as intermediate endpoints for long-term benefit. Torres et al. propose further relevance with reporting of immuno-modulator trials in latent TB, in which adaptive monitoring mitigated adverse immunity induction, a strategy that is directly transferable to pharmacovigilance for long exposure to chemopractive drugs. Real-world integration further depends on electronic health record-linked registries, molecular risk profiling techniques and pharmacoepidemiologic monitoring that needs exceptional sensitivity for toxicity, adherence and cost-effectiveness. Together, adaptive trial designs coupled with real world evidence networks enable rapid discovery of efficacious drug-pathway combinations, maximization of benefit/risk, and approval by regulatory agencies for preventive use among high-risk populations.

LIMITATION

Current evidence for drug repurposing for chemoprevention suffers from limitations of heterogeneity in the design of clinical trials, heterogeneity in biomarker validation and incomplete translational data for phytochemical substances and immuno-modulators [2,20,28]. Although these medications have previously been used to treat other indications, such as prevention, chronic COX-2 inhibition or statin treatment, safety in this scenario is unclear, which makes it difficult to establish optimal dose approach for different populations [8,29,30].

FUTURE SCOPE

Adaptive platform trials involving genomic risk stratification, ctDNA monitoring, and real-world data should be available for rapid validation of repurposed agents in the future [21,25,31,32]. Combination therapy with phytochemicals, immunomodulators and diet-based strategies has promise for synergistic chemoprevention. Plant-derived functional compounds may additionally benefit from bioavailability improvement of nanotechnology-based formulation to have much more translational impact [14,33,34].

CONCLUSION

Cancer chemoprevention is a feasible and mechanistically based approach. Experiments with aspirin, statins, SERMs, and phytochemicals have shown to be beneficial approaches for targeting the AMPK, COX-2, HMG-CoA, and NF-kB signal pathways to inhibit premalignant progress. Precision in identifying phenotypes likely to respond, through validated biomarkers, adaptive trials, and real-world monitoring. By linking mechanistic knowledge with clinical feasibility, repurposed agents have the potential to drive scalable, affordable and targeted prevention of cancer.

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